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Original Article

Evaluation of Cervical Vestibular Evoked Myogenic Potential in Subjects with Chronic Noise Exposure

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OBJECTIVE: Noise has been recognized as a major cause of cochlear damage resulting in both tinnitus and hearing loss. On the other hand, damage to the vestibular system, especially the saccule, can be considered as a potential problem. The cervical vestibular-evoked myogenic potentials (cVEMPs) have been established as a clinical test of measuring both saccular and inferior vestibular nerve function. Therefore, it is thought to be sensitive to the noise-induced damage to the vestibular system. Accordingly, this study was designed to assess the vestibular system in subjects exposed to noise during work by using cVEMPs.

MATERIALS and METHODS: This study was performed in over 60 adult males who were divided into a study group (consisting of 40 adult males) with history of chronic occupational noise exposure and with variable degree of hearing levels and a control group consisting of 20 healthy adults with normal peripheral hearing, with no history of noise exposure and no vestibular complaints. cVEMP recordings were elicited using 95dB nHL click stimuli.

RESULTS: There was statistically significant prolonged cVEMP latency of the P13 and N23 waves of the study versus the control groups. As regard to the sense of imbalance, there were significant prolonged cVEMPs latencies in present versus absent sense of imbalance. However, there were statistically insignificant reduced cVEMP amplitudes in present versus absent sense of imbalance.

CONCLUSION: Chronic noise exposure damages the vestibular system especially the saccule in addition to cochlear damage.

KEYWORDS: Cervical vestibular-evoked myogenic potentials, noise-induced hearing loss, vestibular system

INTRODUCTION

The vestibular system helps in maintaining the balance in association with both the ocular and the central nervous systems. Both the vestibular end organs and the cochlea have a common origin and also utilize the same principle of mechano-electric transduction with the help of the sensory hair cells [1]. Studies on vestibular-evoked myogenic potential (VEMP) have shown that the saccule can be stimulated with loud sound at or above 100dB SPL [2]. Considering this, we can expect that the levels of noise that could cause damage to the cochlea may also damage the vestibular system. Moreover, the studies showed that with similar stimulations the saccule can withstand much lesser force (0.57 gf/mm) than the Reissener's membrane (0.84 gf/mm), implying that the probability of the vestibular system affection due to noise exposure is more than that of the cochlea [3, 4].

Animal studies showed that after exposure to intense noise, there is a pathologic evidence of damage in the utricle, saccule, and semicircular canals. There is also a strikingly resemblance pattern between the damage observed in the cochlea and that observed in the vestibular structures [5]. Two mechanisms are involved in the pattern of destruction of the vestibular end organs by the effect of noise: the first is direct mechanical destruction, whereas the second is metabolic decompensation resulting in degeneration of the sensory elements. So, it is more likely that subjects who have noise-induced hearing loss (NIHL), in addition to the cochlear lesion, will also have damage to the vestibular end organs [6].

Most of the previous studies in humans have measured the horizontal semicircular canal function after prolonged noise exposure [7-9]; however, several histological studies in animals have provided that the saccule may be more susceptible to noise-related damage compared with other vestibular organs [10, 11]. Studies by Perez et al. [12] determined that in rats, the exposure to noise could result in

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changes in the linear (otolith) vestibular-evoked potential differently, whereas the angular (semicircular canal) vestibular-evoked potential still remained unchanged.

Because this type of evoked potential is difficult to be recorded in humans, cervical vestibular-evoked myogenic potentials (cVEMPs) may provide a more comfortable and reliable method to measure the effect of noise in the otolith function. Accordingly, this study was designed to assess the vestibular system in subjects exposed to noise during work by using cVEMPs. So, the aims of this study were: (1) to assess the vestibular system in subjects exposed to noise during work and those having variable hearing thresholds by using cVEMP and (2) to compare cVEMP results in chronic noise exposure subjects and normal subjects.

MATERIALS and METHODS

This study was performed after fulfilling the requirements of our ORL Department Ethical Committee and was approved by the Institutional Research Board of Faculty of Medicine in our university. All participants filled an informed consent form.

This is a prospective cohort study conducted at Audiology Unit, Otolaryngology Department in our hospital. The study was performed from January 2014 to September 2016. The study was performed in over 60 adult males, age ranging from 25 to 55 years. Subjects were divided into two groups. (i) The study group which consisted of 40 adult subjects, aged from 25 to 55 years, with history of exposure to machinery noise during work (from Spinning and Weaving factory exposed to machinery noise for 8 h/day for 6 days a week) and with variable degree of hearing levels. All subjects of the study group had normal middle ear functions.

According to the different hearing thresholds, the study group was further subdivided into four subgroups: The first two subgroups were the short-exposure subgroups (those exposed to noise for duration not exceeding 5 years). The first subgroup (sub-GP1) consisted of 13 adult males who did not have hearing loss at any of the frequency (250-8000 Hz), whereas the second subgroup (sub-GP2) included seven adult males complaining of mild hearing loss only at 4 kHz. The other two subgroups (third and fourth subgroups) were the long-exposure subgroups (those exposed to noise for duration more than 10 years). In the third subgroup (sub-GP3), there were nine adult males complaining of moderate hearing loss only at 4 kHz, whereas the fourth (sub-GP4) consisted of 11 adult males complaining of hearing loss extended to frequencies more than 4 kHz.

(ii) The control group consisted of 20 healthy volunteer adults with normal hearing thresholds in the frequency range of (250-8000 Hz) (hearing threshold level ≤ 2 dB nHL) and bilateral normal middle ear function. All subjects of this group had no history of noise exposure or vestibular complaints.

All subjects in this study were subjected to the following procedures: Full history taking: personal history, history to exclude any otological, neuro-otological diseases, or other systemic disorder. Clinical examination: complete otoscopic examination to ensure patent external auditory canal, no occluding wax, and normal tympanic membrane. (i) Basic audiological evaluation: a) Audiometry (pure tone audiometry at 250, 500, 1000, 2000, 4000, 8000 Hz and speech audiometry including speech reception threshold and speech discrimination) using two channels audiometer, Orbiter 922, Madsen Electronic, version 2 (Denmark). (b) Immittance meter (tympanometry and acoustic reflex) using GSI, tympstar, middle ear analyzer version 2 with 226 Hz probe tone frequency (USA). (ii) cVEMPs using Biologic Auditory Evoked Potential, Navigator Pro, version 7.2.1 ((Natus Medical, Inc., San Carlos, CA, USA). Five electrodes were used for recording of the cVEMPs. Two active electrodes were placed on the middle third of the tonically contracted sternocleidomastoid muscle on each side and two reference electrodes were placed on the middle third of both the clavicles. The ground electrode was placed over the forehead.

During recording, the subject was asked to sit upright while rotating his head to the opposite side of recording and flexing his head about 30 degrees forward to ensure sufficient muscular contraction. Stimulus parameters were 95dB nHL click stimuli of 128 Sweeps and 5/s as a repetition rate delivered by insert earphones. Recording parameters included: (a) the filter settings 30-1500 Hz and (b) the time window was 0-100 ms and with 5.000 gain factor.

Analysis of the Waves

At least two consecutive averages were recorded from each side to verify reproducibility, and the positive and the negative peaks were identified according to their latencies, followed by measuring the amplitude of the wave from base to the peak.

Statistical Analysis

Data were analyzed using Statistical Package for Social Sciences version 22 (IBM Corp.; Armonk, NY, USA). Quantitative parametric data were presented in mean and standard deviation, whereas quantitative non-parametric data were presented in median and 25^{th} and 75^{th} percentiles. The Student's t-test was used for comparing two different groups (parametric data), whereas the Mann-Whitney was used or comparing two different groups (non-parametric data) and Kruskal-Wallis test followed by pairwise comparisons were used for comparing more than two groups (non-parametric data). P value less than 0.05 was considered statistically significant.

RESULTS

All subjects of the study and control groups are age-matched adult males . The study group age was 32.25±10.8 y, whereas the control group age was 33.25±11.2 y, and there was no significant difference between the two groups (p=0.543).

There was no statistically significant different (p>0.05) between the right and left ear as regard to pure tone air conduction and cVEMP results. So, the statistical analysis was conducted on the basis of the number of ears.

Table 1 shows that there was statistically significant elevated PTA at 4-kHz frequencies in sub-GP1 versus control group and 2, 4, and 8 kHz in sub-GP2 versus control group. There was statistically significant elevated PTA threshold at all tested frequencies in sub-GP3 and sub-GP4 versus control.

In sub-GP4, there were statistically significant elevated PTA threshold at most tested frequencies versus sub-GP1, sub-GP2. In sub-GP3, there was statistically significant elevated air conduction at 2, 4, and 8 Hz versus sub-GP1. In sub-GP2, there was statistically significant elevated PTA threshold at 4 and 8 kHz versus sub-GP1.

Table 1. Comparison among different groups of (PTA) in overall tested frequencies

		Groups					
frequency		**Control**	**Subgroup1**	**Subgroup2**	**Subgroup3**	**Subgroup4**	**p**
250 Hz	Median	10.00	10.00	12.50	17.50[abc]	20.00 [abc]	<0.001*
	Percentile 25	5.00	10.00	10.00	15.00	15.00	
	Percentile 75	15.00	15.00	15.00	20.00	25.00	
500 Hz	Median	10.00	10.00	12.50	20.00 [abc]	20.00 [abc]	<0.001*
	Percentile 25	5.00	10.00	10.00	15.00	15.00	
	Percentile 75	15.00	15.00	15.00	25.00	20.00	
1000 Hz	Median	15.00	15.00	15.00	15.00 [a]	25.00 [abc]	<0.001*
	Percentile 25	5.00	10.00	15.00	15.00	15.00	
	Percentile 75	15.00	15.00	20.00	20.00	25.00	
2000 Hz	Median	10.00	15.00	20.00 [a]	25.00 [ab]	25.00 [ab]	<0.001*
	Percentile 25	7.50	15.00	20.00	20.00	20.00	
	Percentile 75	15.00	15.00	25.00	25.00	30.00	
4000 Hz	Median	10.00	20.00 [a]	35.00 [ab]	40.00 [ab]	42.50 [ab]	<0.001*
	Percentile 25	10.00	20.00	30.00	35.00	35.00	
	Percentile 75	15.00	25.00	45.00	45.00	50.00	
8000 Hz	Median	12.50	15.00	25.00 [ab]	20.00 [ab]	35.00 [abcd]	<0.001*
	Percentile 25	10.00	15.00	15.00	20.00	30.00	
	Percentile 75	17.50	20.00	30.00	20.00	55.00	

*:significance <0.05. Test used: Kruskal-Wallis followed by pairwise comparisons

a: significance relative to control group; b: significance relative to Subgroup1; c: significance relative to Subgroup2; d: significance relative to Subgroup

Table 2. Comparison among different groups for cVEMP

		Groups					
frequency		**Control**	**Subgroup1**	**Subgroup2**	**Subgroup3**	**Subgroup4**	**p**
p13 latency	Median	11.84	13.50[a]	13.45[a]	14.30[ac]	15.40[abc]	<0.001*
	Percentile 25	11.10	12.60	12.81	13.40	15.06	
	Percentile 75	13.20	14.60	15.40	15.80	16.00	
p13 amplitude	Median	30.37	19.35[a]	18.00[a]	20.35[a]	14.95[ab]	<0.001*
	Percentile 25	24.60	16.80	16.70	17.15	13.95	
	Percentile 75	35.64	25.00	20.29	22.75	19.40	
n23 latency	Median	18.45	20.43[a]	19.95[a]	21.50[ac]	22.65[abc]	<0.001*
	Percentile 25	17.75	19.60	19.22	19.95	22.15	
	Percentile 75	19.60	21.80	21.50	22.60	22.80	
n23 amplitude	Median	20.40	15.65[a]	14.70[a]	16.00[a]	13.70[a]	<0.001*
	Percentile 25	19.05	14.50	14.45	14.95	12.75	
	Percentile 75	23.40	17.80	17.55	17.95	17.00	

*:significance <0.05. Test used: Kruskal-Wallis followed by pairwise comparisons

a: significance relative to control group; b: significance relative to Subgroup1; c: significance relative to Subgroup2; d: significance relative to Subgroup3

Two adults with absent cVEMP (i.e., four ears) in sub-GP3 and four adults with absent cVEMP (i.e., eight ears) in sub-GP4 were not included in Table 2.

Table 2 also shows that there was statistically significant prolonged cVEMP latency of the P13 and N23 waves of the different subgroups (1-2-3 and 4) versus the control group. As regard to the relation between cVEMP latency in the different subgroups, there was statistically significant prolonged latency of theP13 and N23 waves of the sub-G4 versus sub-GP1 and sub-GP2. Also, there was statistically significant prolonged latency of the P13 and N23 waves of the sub-GP3 versus sub-GP2. As regard to cVEMP amplitude, there was statistically significant reduction of the P13 and N23 amplitude in all subgroups versus control group.

There were statistically significant prolonged cVEMP latencies in present versus absent sense of imbalance. However, there were statistically insignificant reduced cVEMP amplitudes in present versus

Table 3. Comparison between the present and absent sense of imbalance and cVEMP in study group

		Sense of imbalance		
		Absent	Present	p
p13 latency	Median	13.10	15.16	<0.001*
	Percentile 25	12.60	14.40	
	Percentile 75	13.27	15.80	
p13 amplitude	Median	19.35	18.50	>0.05
	Percentile 25	15.80	14.40	
	Percentile 75	23.42	20.80	
n23 latency	Median	19.50	21.90	<0.001*
	Percentile 25	19.02	20.40	
	Percentile 75	20.70	22.80	
n23 amplitude	Median	15.50	14.10	>0.05
	Percentile 25	14.50	12.20	
	Percentile 75	17.45	19.50	

*:significance <0.05. Test used: Mann-Whitney test

absent sense of imbalance as shown in Table 3. There were six adults who had absent cVEMP waves and were not included in this table.

DISCUSSION

It is clear that prolonged duration of exposure to occupational noise causes permanent hearing damage. The characteristic finding of noise-induced loss is high-frequency hearing loss with the characteristic 4-kHz notch. The mechanism of NIHL is related to some cellular changes that occur in the inner ear. These changes may be due to either a direct mechanical trauma or metabolic changes resulting in ischemia, reactive oxygen radicals causing metabolic overload in the organ of Corti, in early stages of NIHL. Almost about 30 dB hearing loss at 4 kHz frequency can be seen, and this loss is caused by damage of its related place in the organ of Corti. The histopathological studies have shown that noise exposure causes damage to the cochlea specifically at a 9- to 13-mm area of the cochlea. This place is responsible for the 4-kHz frequency response [13].

The current study showed that as the duration of hearing loss was increased, the hearing loss progressed to frequencies other than 4 kHz and a higher frequency hearing loss occurred followed by hearing loss that extend to lower frequencies with flattening of the audiometric notch to the degree that the audiogram could slope downward at frequencies as low as 0.5 kHz, as reported by Hong [14].

In the present study, the cVEMP latency (P13-N23 latency) was increased in all of the different study subgroups versus the control group and also the cVEMP latency (P13-N23 latency) was increased between the different subgroups in relation to each other, where the sub-GP4 (the subgroup with the severe hearing loss) showed the most delayed latency as compared with sub-GP1 (where there was no hearing loss), sub-GP2 and sub-GP3 (in both of these subgroups, the hearing loss is limited only to 4 KHz); also in the different subgroups, as the severity and duration of hearing loss are increased, the cVEMP latency was increased. The cVEMP amplitude (P13-N23 amplitude) showed statistically significant reduction of the P13-N23 amplitude in relation to the control group.

cVEMP latency results were in agreement with Kumar et al. [15], they reported that in NIHL, as the pure tone average increased, the latency was prolonged, also our results are in agreement with Wang and Young [16] who reported that an increasingly damaged saccule could result in abnormal VEMPs (e.g., absent or delayed VEMPs) in subjects with >40dB at 4 kHz. Similar results are also obtained by Tseng and Young [17] who reported that there was a decreasing order of abnormal percentages in the function of the cochlea, saccule, utricle, and semicircular canals after exposure to a chronic noise, this further supports that pars inferior (cochlea and saccule) is more vulnerable to noise damage than pars superior (utricle and semicircular canals).

It is clear that the cochlea and the vestibular end organs have a common evolutionary origin and can utilize the same principle of mechano-electric transduction with the help of the sensory hair cells. Studies on VEMP have shown that the saccule can be stimulated with loud sound level at or above 100dB SPL. Considering this, the noise levels that can cause cochlear damage could also stimulate the vestibular system [3, 4], and these results can explain the abnormal delay of the cVEMPs (P13-N23 latency) in sub-GP1 (normal hearing subgroup in relation to the control group). The results of the current study are also strongly supplemented by Raghunath et al. [18]; they found that the vestibular deficits were prevalent prior to clinically evident hearing loss.

Recent researches by Vetter [19] about the cellular signaling protective mechanism against NIHL and the role for some novel intrinsic cochlear signaling such as corticotropin-releasing factor the cellular signaling processes, which occurs in the cochlea, is believed to be involved in protection against the NIHL. According to Vetter [19], these protective mechanisms have never been assessed as protective mechanism of the vestibular end organs again, this may raise our probability of the vestibular affection before the cochlear affection with chronic noise exposure.

This study concluded that there was a significant decrease in the cVEMP amplitude in relation to the control group, similar results were obtained by Kumar et al. [15]; while these results disagree with Emara and Gabr [20] who reported that there was statistically insignificant reduction of the cVEMP amplitude with increase of the duration and the degree of hearing loss.

According to Alpini et al. [21], the amplitude of the (P13-N23) is highly variable among subjects or even in one subject between trials and between methods used to elicit the response and they attributed this variability to lack of cooperation from participant, the presence of neck stiffness and inability to keep the SCM tonically contracted for long time.

In the present study, the comparison between the subjective imbalance sensation and the cVEMP results revealed statistically significant delayed response to those with imbalance sensation versus subjects without imbalance sensation. The results of this study were agreed with Dalgıç et al. [13] who found more cVEMP abnormality in subjects complaining of vertigo in relation to those without vertigo. Similar results reported by Wang and Young [16] and Sazgar et al. [6] showed that there was a relation between vertigo and the delayed cVEMP latency after exposure to acute acoustic trauma.

The results of this study were suggestive that the sacculocollic pathway may be more susceptible to noise-related damage; Hsu et al. [22] correlated the loss of cVEMPs after long-term noise exposure in guinea pigs to some morphological changes in the saccule. Specifically, the cell bodies in the hair cells of the saccular maculae showed signs of atrophy and disruption in the guinea pigs with post-noise exposure cVEMP loss. This explanation may be used in our cases.

CONCLUSION

Chronic noise exposure damages the vestibular system especially the saccule in addition to cochlear damage.

Ethics Committee Approval: This study was approved by the Institutional Research Board of Faculty of Medicine, Mansoura University (Approval Date:19.01.2015/Approval No: MS/838).

Informed Consent: Written informed consent was obtained by the patients who participated in this study.

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REFERENCES

1. Halmagyi GM, McGarvie LA, Aw ST, Yavor RA, Todd MJ. The click-evoked vestibulo-ocular reflex in superior semicircular canal dehiscence. Neurology 2003; 60: 1172-5. [CrossRef]
2. Minor LB. Clinical manifestations of superior semicircular canal dehiscence. Laryngoscope 2005; 115: 1717-27. [CrossRef]
3. Halmagyi GM, Curthoys IS. Clinical testing of otolith function. Ann N Y Acad Sci 1999; 871: 195-204. [CrossRef]
4. Watson SR, Halmagyi GM, Colebatch JG. Vestibular hypersensitivity to sound (Tullio phenomenon): Structural and functional assessment. Neurology 2000; 54: 722-8. [CrossRef]
5. Wilhelmsen K, Strand LI, Nordahl SHG, Eide GE, Ljunggren AE. Psychometric properties of the Vertigo symptom scale - Short form. BMC Ear Nose Throat Disord 2008; 8: 2. [CrossRef]
6. Sazgar A, Dortaj V, Akrami K, Akrami S, Karimi Yazdi AR. Saccular damage in patients with high-frequency sensorineural hearing loss. Eur Arch Otorhinolaryngol 2006; 263: 608-13. [CrossRef]
7. Man A, Segal S, Naggan L. Vestibular involvement in acoustic trauma (An electronystagmographic study). J Laryngol Otol 1980; 94: 1395-400. [CrossRef]
8. Oosterveld WJ, Polman AR, Schoonheyt J. Noise-induced hearing loss and vestibular dysfunction. Aviat Space Environ Med 1980; 51: 823-6.
9. Golz A, Westerman ST, Westerman LM, Goldenberg D, Netzer A, Wiedmyer T, et al. The effects of noise on the vestibular system. Am J Otolaryngol 2001; 22: 190-6. [CrossRef]
10. McCabe BF, Lawrence M. The Effects of intense sound on the non-auditory labyrinth. Acta Otolaryngol 1958; 49: 147-57. [CrossRef]
11. Mangabeira-Albernaz PL, Covell WP, Eldredge DH. Changes in the vestibular labyrinth with intense sound. Laryngoscope 1959; 69: 1478-93. [CrossRef]
12. Perez R, Freeman S, Cohen D, Sohmer H. Functional impairment of the vestibular end organ resulting from impulse noise exposure. Laryngoscope 2002; 112: 1110-4. [CrossRef]
13. Dalgıç A, Yılmaz O, Hıdır Y, Satar B, Gerek M. Analysis of Vestibular Evoked Myogenic Potentials and Electrocochleography in Noise Induced Hearing Loss. J Int Adv Otol 2015; 11: 127-32. [CrossRef]
14. Hong O. Hearing loss among operating engineers in American construction industry. Int Arch Occup Environ Health 2005; 78: 565-74. [CrossRef]
15. Kumar K, Vivarthini CJ, Bhat JS. Vestibular evoked myogenic potential in noise-induced hearing loss. Noise Health 2010; 12: 191-4. [CrossRef]
16. Wang YP, Young YH. Vestibular evoked myogenic potentials in chronic noise induced hearing loss. Otolaryngol Head Neck Surg 2007; 137: 607-11. [CrossRef]
17. Tseng CC, Young YH. Sequence of vestibular deficits in patients with noise-induced hearing loss. Eur Arch Otorhinolaryngol 2013; 270: 2021-6. [CrossRef]
18. Raghunath G, Suting LB, Maruthy S. Vestibular symptoms in factory workers subjected to noise for a long period. Int J Occup Environ Med 2012; 3: 136-44.
19. Vetter DE. Cellular signaling protective against noise-induced hearing loss-A role for novel intrinsic cochlear signaling involving corticotropin-releasing factor? Biochem Pharmacol 2015; 97: 1-15. [CrossRef]
20. Emara AA, Gabr TA. Chronic noise exposure: impact on the vestibular function. Advanced Arab Academy of Audiovestibulogy Journal 2014; 1: 71-9. [CrossRef]
21. Alpini D, Pugnetti L, Caputo D, Cornelio F, Capobianco S, Cesarani A. Vestibular evoked myogenic potentials in multiple sclerosis: clinical and imaging correlations. Mult Scler 2004; 10: 316-21. [CrossRef]
22. Hsu WC, Wang JD, Lue JH, Day AS, Young YH. Physiological and morphological assessment of the saccule in Guinea pigs after noise exposure. Arch Otolaryngol Head Neck Surg 2008; 134: 1099-106.[CrossRef]